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Reduced Nogo-P3 in adults with developmental coordination disorder (DCD)

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Abstract

Nogo-N2 is associated with the premotor cognitive process that precedes motor response (e.g., conflict monitoring), whereas Nogo-P3 is related to the inhibition of the actual motor response. We examined the influence of motor clumsiness of developmental coordination disorder (DCD) on components of the event-related potential in a Go/Nogo task. Participants were healthy adults (N=81) that were classified into control and DCD groups based on the Movement Assessment Battery for Children Second Edition. We manipulated the difficulty in stopping a response by varying the frequency of Nogo stimuli in a response task into rare (20 %) and frequent (80 %) conditions, and Nogo-N2 and Nogo-P3 were calculated from electroencephalograms (EEGs) during the Go/Nogo tasks. The commission error rate in the rare condition was significantly higher in the DCD group than in the control group, indicating that motor clumsiness decreases task performance. There were no differences in Nogo-N2 between DCD and control groups. However, Nogo-P3 in the rare condition was reduced in the DCD group compared to the control group. These results suggest that the influence of motor clumsiness is limited to the cognitive process after the initiation of the actual motor response.

Keywords: motor clumsy; developmental coordination disorder; inhibition; cognitive control; event-related potentials

1. Introduction

Motor and cognitive processes are known to interact with each other (Diamond, 2000; Middleton and Strick, 2000). The developmental coordination disorder (DCD) is a neurodevelopmental disorder characterized by so-called motor clumsiness, which is defined as a difficulty in the acquisition and execution of coordinated motor skills (American Psychiatric Association, 2013; World Health Organization, 2019). For example, in people with DCD, poor performances are observed in peg board tasks, ball catching, and the one-leg balance (Henderson et al., 2007). People with DCD also have difficulties in performing cognitive tasks (Wilson et al., 2013). This study focused on inhibition tasks, such as the Go/Nogo task, in which participants are asked to respond to a Go stimulus and to stop the response to a Nogo stimulus. He et al. (2018) reported that adults with DCD performed poorly in the Go/Nogo task compared to adults with typical development (TD). Identical results were obtained for other inhibition tasks such as emotional face Go/Nogo task (Rahimi-Golkhandan et al., 2016; Rahimi-Golkhandan et al., 2015), the stop signal task (He et al., 2018), the Simon task (Mandich et al., 2002), and the Stroop task (Pratt et al., 2014). These results indicated that motor clumsiness decreases the performance of inhibition tasks.

Several motor and cognitive processes are known to be involved in inhibition tasks (Nakata et al., 2015). The event-related potentials (ERPs), which can be measured by electroencephalograms (EEGs), are a useful tool for examining temporal characteristics of information processing. There are two well-known ERP components associated with inhibition tasks: the N2, or the frontal negative ERP component peaking from 200 ms to 300 ms, and the P3 following the N2, which is a positive component with frontocentral to centroparietal topography (Huster et al., 2013). In this study, the ERP components elicited by events requiring response inhibition were labeled Nogo-N2 and Nogo-P3, whereas ERP components elicited by events requiring response execution were labeled Go-N2 and Go-P3. These ERP components have different functional significances (Huster et al., 2013).

It has been reported that Nogo-N2 is larger than Go-N2 (Pfefferbaum et al., 1985), and it is larger when participants successfully stopped a response than when they failed to stop (Schmajuk et al., 2006). Moreover, Nogo-N2 is also enhanced by emphasizing response speed (Jodo and Kayama, 1992), as well as by increasing the muscle force used for making the response (Nakata et al., 2006). These findings suggest that Nogo-N2 might be associated with the inhibition of motor responses. However, there is no difference in Nogo-N2 between individuals with shorter or longer RTs, regardless of the effort required to stop a fast response (Smith et al., 2006). Furthermore, Nogo-N2 is not modulated by the level of response preparation in the cued-Go/NoGo task (Bruin et al., 2001). These findings indicate that Nogo-N2 is unrelated to inhibition of the actual motor response. Gajewski and Falkenstein (2013) suggested that Nogo-N2 is associated with the inhibition of premature response plans. In addition, N2 was enhanced by the stimulus characteristics of the competing response options (Bartholow et al., 2005; Groom and Cragg, 2015; Kopp et al., 1996). Based on these findings, researchers have proposed that Nogo-N2 reflects the degree of the conflict between tendencies to execute or stop a response before executing the response (Botvinick et al., 2001; Stahl and Gibbons, 2007; Yeung et al., 2004). Therefore, we considered that Nogo-N2 is associated with the premotor cognitive process that precedes motor response.

Nogo-P3 has a topography that is more frontal than Go-P3 (Fallgatter and Strik, 1999; Simson et al., 1977). Nogo-P3, unlike Nogo-N2, is larger in people with shorter RTs compared to those with longer RTs (Smith et al., 2006), and it is modulated by the degree of response preparation (Bruin et al., 2001). Moreover, Nogo-P3 is not modulated by the degree of response conflict (Randall and Smith, 2011). Therefore, Nogo-P3 might be related to the inhibition of motor responses (Bruin et al., 2001; Smith et al., 2006). However, the latency of Nogo-P3 is too long to correspond to the inhibition of motor response. Transcranial magnetic stimulation studies have indicated that the inhibitory process begins 140 ms after stimulus presentation (Coxon et al., 2006; Van den Wildenberg et al., 2010), whereas the peak of Nogo-P3 is

approximately 350 ms after stimulus presentation (Huster et al., 2013). Therefore, Nogo-P3 might be associated with the aftereffects of response inhibition, such as the evaluation and/or monitoring of the outcomes of response inhibition (Huster et al., 2013; Schmajuk et al., 2006). Taken together, we considered that Nogo-N2 is associated with the premotor cognitive process that precedes motor response, whereas Nogo-P3 is related to the aftereffects of inhibiting the actual motor response.

Nogo-N2 and Nogo-P3 change differently in people with neurological and psychiatric disorders related to motor function. For example, people with Parkinson's disease have atypical Nogo-N2 and Nogo-P3 (Beste et al., 2010), whereas the effect of Huntington's disease has been reported only on Nogo-P3 (Beste et al., 2008; Beste et al., 2010). Moreover, children with attention deficit hyperactivity disorder (ADHD) tend to display motor clumsiness (Pitcher et al., 2003), and a smaller Nogo-N2 has been reported in children with ADHD than those with typical development (TD), which was not the case for Nogo-P3 (Inoue et al., 2010). Therefore, we considered that Nogo-N2 and Nogo-P3 were differently modulated by motor clumsiness in DCD.

This study was designed to examine the characteristics of Nogo-N2 and Nogo-P3 in adults with DCD. The motor clumsiness of adults was evaluated using the Movement Assessment Battery for Children Second Edition (MABC-2; Henderson et al., 2007; Hirata et al., 2018; Kita et al., 2016) which is a familiar tool for assessing DCD (Blank et al., 2012) because there is no suitable test for adults (Wilmut and Byrne, 2014). We manipulated the level of difficulty in stopping a response by varying the frequency of Nogo stimuli in a Go/Nogo task (Nieuwenhuis et al., 2003) by assuming that it would be more difficult to stop responding to rare Nogo stimuli than to frequent ones. We predicted that rare Nogo stimuli compared to frequent ones would more strongly enhance Nogo-N2 and Nogo-P3 and that the differences in ERP components between groups would be significantly larger for rare Nogo stimuli.

2. Methods

2.1. Participants

Participants were healthy adults (N =97). Two of the participants that did not complete EEG recordings, one participant with a high commission error rate in the rare Nogo condition (37.5%, Others: < 30%), four participants with recording problems, and nine participants with excessive artifact was excluded, and data of 81 participants were used for the analysis. MABC-2 Age-Band 3 (11 – 16 years: (Henderson et al., 2007) was used for the assessment of motor clumsiness. Participants whose total score was 7 or less (i.e., ≦1SD) were classified as the DCD group, and the others were the control group (Table 1). There were no differences between groups with regard to age, sex, handedness, and the Wender Utah Rating Scale score (Matsumoto et al., 2005; Ward, 1993). Some of these data have been submitted for publication in a different study (Kita et al., in submission). The protocol of this study was approved by the ethics committee at the National Center of Neurology and Psychiatry (NCNP) (approval number A2017-027).

2.2. Task

The experimental stimuli consisted of red or blue circles (width = 4.52°) that were presented on grey backgrounds for 100 ms (Figure 1). The interstimulus intervals were set at 1300 or 1500 ms. Participants were informed that a blue circle was a Go stimulus and a red circle was a Nogo stimulus, and were required to press a button with the thumb of their dominant hand for a Go stimulus, and to refrain from responding to a Nogo stimulus. The tasks were performed under two conditions with different frequencies of Nogo stimuli set at either 80% (frequent Go stimuli) or 20% (rare Nogo stimuli) and 20% (rare Go stimuli) or 80% (frequent Nogo stimuli). One block included 160 trials, and two blocks were performed for each condition. The order of

presenting the conditions was counterbalanced among participants. The task was administered on a PC monitor using E-Prime 2.0 (Psychology Software Tools, Inc.).

2.3. Electroencephalogram (EEG) recordings and analysis

EEGs and electrooculograms (EOGs) were sampled at 2048 Hz using an Active 2 system (Biosemi, Inc., Amsterdam). Common Mode Sense (CMS) and Driven Right Leg (DRL) electrodes were placed on AF1 and AF2. EEGs were recorded from 32 scalp sites (i.e., Fp1, Fpz, Fp2, F7, F3, Fz, F4, F8, FC3, FCz, FC4, T7, C3, Cz, C4, T8, CP3, CPz, CP4, P7, P3, Pz, P4, P8, PO7, PO3, POz, PO4, PO8, O1, Oz, O2), and EOGs were recorded from electrodes above and at the outer canthus of the left eye.

EEG and ERP were analyzed using EEGLAB 15 (Delorme and Makeig, 2004) in MATLAB 2010Ra (Mathworks Inc., Natick, MA, USA). Offline data were down-sampled at 1000Hz, and band-pass filtered at 0.1-40Hz. Stimulus-locked epochs were extracted from −100 to 800 ms, in which -100-0 ms was used as the baseline. Artifacts related to eye movement were corrected based on principal component analysis using Automatic Artifact Removal toolbox v1.3 (Gómez-Herrero, 2007). Then, EOG channels were excluded, and EEGs were re-referenced to a common average reference. We excluded epochs in which the activity exceeded ±70 μV, and epochs, including artifacts, by visual inspection. The ERP was computed from epochs for Go and Nogo stimuli under each condition. We used ERP amplitudes of N2 and P3 for the statistical analysis. N2 amplitudes were calculated as the mean voltage of the 100 ms period from 200 to 300 ms, and P3 amplitudes were calculated as the mean voltage of the 100 ms period from 300 to 400 ms.

2.4. Statistical analysis

For the correct RT, the omission error rate, and the commission error rate, we conducted analyses of variance (ANOVAs) with group (control and DCD) and condition (rare Nogo and

frequent Nogo) as the independent variables. We used the amplitudes at Fz and Cz for the analyses of Nogo-N2 and Go-N2 because N2 is maximum at frontal and central electrodes (Huster et al., 2013), whereas the amplitudes at Fz, Cz, and Pz were used for Nogo-P3 and Go-P3 analyses because P3 is broadly distributed (Huster et al., 2013). We conducted ANOVAs with group, electrode, and stimulus (rare, frequent) for the ERP amplitudes of Nogo-N2, Nogo-P3, Go-N2, and Go-P3. The degree of freedom was corrected by Greenhouse-Geisser procedure when appropriate.

3. Results

3.1. Behavioral results

Table 2 shows the behavioral results in the DCD and control groups. The results for the correct RT indicated a significant main effect of condition ($F(1,79) = 221.05$, $p < .001$, $\eta_p^2 = .74$), whereas there was neither a significant effect of group ($F(1,79) = 0.38$, $p = .54$, $\eta_p^2 = .005$) nor a group by condition interaction ($F(1,79) = 3.40$, $p = .07$, $\eta_p^2 = .04$). Moreover, the results of the omission error rate indicated neither a significant main effects of group ($F(1,79) = 0.05$, $p = .83$, $\eta_p^2 = .0006$) nor condition ($F(1,79) = 0.02$, $p = .88$, $\eta_p^2 = .0003$), whereas there was a significant interaction between group and condition ($F(1,79) = 4.56$, $p = .03$, $\eta_p^2 = .05$); however, a simple effect analyses showed no significant effects of group (rare: $F(1,79) = 1.26$, $p = .03$, $\eta_p^2 = .02$; frequent: $F(1,79) = 0.90$, $p = .35$, $\eta_p^2 = .01$) and condition (control: $F(1,79) = 3.73$, $p = .06$, $\eta_p^2 = .06$; DCD: $F(1,79) = 1.42$, $p = .24$, $\eta_p^2 = .06$). Also, the results of the commission error rate indicated a significant main effect of group ($F(1,79) = 5.08$, $p = .02$, $\eta_p^2 = .06$) and condition ($F(1,79) = 112.37$, $p < .001$, $\eta_p^2 = .58$), and a significant interaction between them ($F(1,79) = 5.28$, $p = .02$, $\eta_p^2 = .06$). Simple effect analyses indicated a significant main effect of group in the rare Nogo condition ($F(1,79) = 5.20$, $p = .03$, $\eta_p^2 = .06$), whereas there was no significant main effect of group in the frequent Nogo condition ($F(1,79) = 0.01$, $p = .75$, $\eta_p^2 = .001$). These behavioral results indicated a shorter correct RT for the rare Nogo than for the frequent Nogo

condition, a higher commission error rate for the rare Nogo than for the frequent Nogo condition, and a higher commission error rate for the rare Nogo condition in the DCD group than in the control group.

3.2. Event related potential (ERP)

Figure 2 shows the ERP waveforms for Go and Nogo stimuli for each condition in the control and the DCD groups. Figure 3 shows the scalp topographies of ERP components for each condition in the control and DCD groups. We observed that all ERP components were larger for the rare than for the frequent stimuli (Figure 2 and Table 3). Moreover, Nogo-P3 was distributed in the central area, whereas Go-P3 was distributed in the parietal area (Figure 3). Nogo-P3 seemed smaller in the DCD than in the control group (Figure 2 and Table 3).

3.2.1. Nogo N2

There were significant main effects of electrode ($F(1,79) = 17.88$, $p < .001$, $\eta_p^2 = .18$) and stimulus ($F(1,79) = 59.51$, $p = .001$, $\eta_p^2 = .43$) and a significant interaction between them ($F(1,79) = 7.78$, $p < .01$, $\eta_p^2 = .09$). Simple effect analysis showed no significant effects of electrode in the rare stimulus ($F(1,79) = 2.12$, $p = .15$, $\eta_p^2 = .03$), whereas the other main effects were significant (stimulus at Fz: $F(1,79) = 38.04$, $p < .001$, $\eta_p^2 = .32$; stimulus at Cz: $F(1,79) = 53.74$, $p < .001$, $\eta_p^2 = .40$; electrode at frequent stimulus: $F(1,79) = 56.57$, $p < .001$, $\eta_p^2 = .42$).

3.2.2. Nogo P3

There were significant main effects of electrode ($F(2,158) = 58.67$, $p < .001$, $\eta_p^2 = .43$, $\varepsilon = .91$) and stimulus ($F(1,79) = 96.91$, $p < .001$, $\eta_p^2 = .55$) and a significant interaction between them ($F(2,158) = 45.50$, $p < .001$, $\eta_p^2 = .37$). Simple effect analysis showed that all pairs of main effects were significant (stimulus at Fz: $F(1,79) = 16.99$, $p < .001$, $\eta_p^2 = .18$; stimulus at Cz: $F(1,79) = 112.06$, $p < .001$, $\eta_p^2 = .59$; stimulus at Pz: $F(1,79) = 129.58$, $p < .001$, $\eta_p^2 = .62$;

electrodes at rare stimulus: $F(2,158) = 67.53$, $p < .001$, $\eta_p^2 = .46$; electrodes at frequent stimulus: $F(2,158) = 15.83$, $p < .001$, $\eta_p^2 = .17$, $\varepsilon = .89$). Bonferroni post hoc tests showed that the P3 amplitude for the rare stimulus was the largest at Cz, and the P3 amplitude at Pz was larger than that at Fz (all *ps* < .001). The tests showed that the P3 amplitudes at Cz and Fz for the frequent stimulus were larger than that at Fz (*ps* < .001), but these differences were not significant ($p > .05$). Crucially, we found a significant interaction between group and stimulus ($F(1,79) = 5.38$, $p = .02$, $\eta_p^2 = .06$). A simple effect analysis showed that Nogo-P3 amplitudes in the DCD group were smaller than those in the control group for the rare stimulus ($F(1,79) = 4.03$, $p = .048$, $\eta_p^2 = .05$), whereas this difference was not significant for the frequent stimulus ($F(1,79) = 0.06$, $p = .80$, $\eta_p^2 = .0008$).

3.2.3. Go N2

There was a significant main effect of electrode ($F(1,79) = 50.17$, $p < .001$, $\eta_p^2 = .39$), a significant interaction between group and stimulus ($F(1,79) = 5.91$, $p = .02$, $\eta_p^2 = .07$), and a significant interaction between electrode and stimulus ($F(1,79) = 29.55$, $p < .001$, $\eta_p^2 = .27$). Regarding to the interaction between group and stimulus, we also found a significant main effect of stimulus in the control group ($F(1,55) = 4.25$, $p = .04$, $\eta_p^2 = .07$), whereas the main effect was not significant in the DCD group ($F(1,24) = 7.17$, $p = .13$, $\eta_p^2 = .09$). A simple effect analysis indicated that all main effects pairs in the significant interaction between electrode and stimulus were significant (stimulus at Fz: $F(1,79) = 5.58$, $p < .05$, $\eta_p^2 = .07$; stimulus at Cz: $F(1,79) = 5.00$, $p < .05$, $\eta_p^2 = .06$; electrode at rare stimulus: $F(1,79) = 14.53$, $p < .001$, $\eta_p^2 = .16$; electrode at frequent stimulus: $F(1,79) = 92.10$, $p < .001$, $\eta_p^2 = .54$).

3.2.4. Go P3

We found significant main effects of electrode ($F(2,158) = 118.51$, $p < .001$, $\eta_p^2 = .60$, GG $\varepsilon = .83$) and stimulus ($F(1,79) = 31.99$, $p < .001$, $\eta_p^2 = .29$), and a significant interaction

between them ($F(2,158) = 38.60$, $p < .001$, $\eta_p^2 = .33$, $\varepsilon = .80$). A simple effect analysis indicated a significant main effect of stimulus at Pz ($F(1,79) = 169.97$, $p < .001$, $\eta_p^2 = .68$), whereas there was neither a significant main effect of stimulus at Fz ($F(1,79) = 0.23$, $p = .63$, $\eta_p^2 = .003$) nor Cz ($F(1,79) = 3.03$, $p = .08$, $\eta_p^2 = .004$). A simple effect analysis indicated a significant main effect of electrode in the rare ($F(1,79) = 100.91$, $p < .001$, $\eta_p^2 = .56$) and frequent stimuli ($F(1,79) = 85.87$, $p < .001$, $\eta_p^2 = .52$). Bonferroni post hoc tests showed that Go-P3 amplitude at Pz was larger than at Fz and Cz for the rare stimulus, whereas Go-P3 amplitude at Cz and Pz were larger than at Fz (all *ps* < .001).

4. Discussion

We examined the characteristics of Nogo-N2 and Nogo-P3 in adults with DCD by manipulating the level of difficulty in stopping a response by varying the frequency of Nogo stimuli. As expected, the correct RT was shorter in the rare Nogo condition than in the frequent Nogo condition, and there was a higher commission error rate in the rare Nogo condition than the frequent Nogo condition. These results indicated that it was more difficult to stop a response for rare Nogo stimuli than frequent ones. Consistent with previous studies (Nieuwenhuis et al., 2003; Ramautar et al., 2004), we also found rare Nogo stimuli more strongly enhanced Nogo-N2 and Nogo-P3 than frequent Nogo stimuli. These results confirmed that Nogo-N2 and Nogo-P3 were modulated by the level of difficulty in stopping a response in the Go/Nogo task.

Comparing the control and the DCD group in the Go/Nogo task indicated that the DCD group had a higher commission error rate for the rare Nogo condition than the control group, whereas there were no differences between groups in the correct RT and the commission error rate. He et al. (2018) reported that the correct RT was positively correlated with the accuracy in the Go/Nogo task, and the accuracy adjusted by the correct RT was lower in adults with DCD than those with TD. These findings suggest that people with DCD have difficulties in stopping the motor response.

No differences were found between the DCD group and the control group with regards to Nogo-N2, but Nogo-P3 for the rare stimulus was smaller in the DCD group than in the control group. Previous studies have suggested that Nogo-N2 is associated with the premotor cognitive process that precedes motor response (Gajewski and Falkenstein, 2013; Yeung et al., 2004) , and Nogo-P3 reflects the aftereffects of the inhibition of the actual motor response (Bruin et al., 2001; Smith et al., 2006). These results indicate that the influence of motor clumsiness is limited to the cognitive process after the initiation of the actual motor response in the Go/Nogo task. Tsai et al. (2009) reported that P3 amplitude on the Posner cueing task was smaller in children with DCD than in those with TD. In addition, latency from N2 to RT was longer in children with DCD than those with TD, but there were no differences between them in N2 latency and amplitude (Tsai et al., 2009). Therefore, we suggested that people with DCD exhibit impaired initiation of the actual motor response and the subsequent cognitive process.

In previous studies, children with DCD committed more errors in the context of the Nogo stimulus of happy face than children with TD did, but no difference was found for the Nogo stimuli of neutral, sad, and fearful faces (Rahimi-Golkhandan et al., 2016; Rahimi-Golkhandan et al., 2015). This finding suggested that children with DCD are more susceptible to the happy face stimulus than the children with TD, leading to decreased performance in inhibition tasks. Zhang and Lu (2012) reported that positive and negative faces enhanced Nogo-P3 to a greater degree than the neutral face, but no enhancement was found for Nogo-N2. In addition, Nogo-P3 was larger for the positive context than the neutral and negative contexts, but Nogo-N2 was not modulated by emotional context (Albert et al., 2010). Hence, previous studies have indicated that emotional effects occur only for Nogo-P3, which implies that the decrease in performance for happy face in children with DCD (Rahimi-Golkhandan et al., 2016; Rahimi-Golkhandan et al., 2015) could be related to Nogo-P3. These findings supported our results showing that the influence of motor clumsiness is limited to Nogo-P3.

The Go/Nogo task is combined with other tasks to form executive function tasks, involving higher-order cognitive processes such as working memory, inhibition, and cognitive flexibility (Diamond, 2013). These tasks are used to investigate characteristics in people with developmental disorders in a more comprehensive way (Barkley, 1997; Ozonoff, 1995). Executive function tasks can be classified into three groups: visuospatial tasks, verbal tasks (i.e., verbal comprehension or verbal response), and motor tasks (Leonard and Hill, 2015). Previous studies have found poor performance among people with DCD on visuospatial and motor tasks, but performance was not remarkable on verbal tasks (Leonard et al., 2015; Leonard and Hill, 2015). In this study, we found it difficult to stop the motor response in the DCD group, and difficulties were associated with the cognitive process after the initiation of the actual motor response. The poor performance of people with DCD on motor tasks may be rooted in cognitive processes that occur after the beginning of the motor response, but visuospatial tasks may be associated with other mechanisms.

In a study similar to ours, Nogo-P3 was found to be lower in individuals with Huntington's disease than in healthy controls, but no difference in Nogo-N2 was found between them (Beste et al., 2008; Beste et al., 2010). Beste et al. (2010) reported reductions in Nogo-N2 and Nogo-P3 in individuals with Parkinson's disease. In relation to the differences in pathology between these diseases, Beste et al. (2010) suggested that Nogo-N2 is related to the nigro-striatal dopamine system, while Nogo-P3 is related to the mesocortico-limbic dopamine system. In a review, the basal ganglia was suggested as a neural signature of DCD (Biotteau et al., 2016). Thus, it is possible that clumsiness in DCD could be related to the mesocortico-limbic dopamine system.

Two functional magnetic resonance imaging studies have explored the characteristics of children with DCD in the Go/Nogo task. In one study, path coefficients from the anterior cingulate cortex (ACC) to inferior parietal cortex increased in children with DCD compared to those with TD, suggesting that ACC was more actively involved when performing the Go/Nogo

task by children with DCD than those with TD (Querne et al., 2008). On the other hand, the present study suggested that the activity of ACC might be reduced in the DCD group compared to the control group, because ACC is one of the candidate generators of Nogo-P3 (Beste et al., 2008). In the other study, the different activations were not found between children with DCD and those with TD, whereas the activity of primary and sensory cortex decreased in children with co-occurring DCD and ADHD (Thornton et al., 2018). In the present study, there were no differences in ADHD traits between the DCD group and the control group. These inconsistencies might be caused by differences in the temporal resolution and the age-range. However, the fMRI studies had a small sample (Querne et al., 2008; Thornton et al., 2018), and moreover, the findings on ERP and fMRI related to DCD are insufficient. Therefore, it is suggested that future studies using neurophysiological measures should explore the neural basis of DCD.

Previous studies have reported differences in ERP components unrelated to motor response between people with DCD and those with TD. In a passive auditory oddball task, P3 amplitude was lower in children with DCD than in children with TD, and mismatch negativity was not observed for children with DCD (Holeckova et al., 2014). In the Posner cuing task, N1 latency was longer in children with DCD than in children with TD (Tsai et al., 2009). Although our results found only a limited influence of motor clumsiness after the initiation of the motor response, it is possible that attentional and premotor processes are influenced by motor clumsiness in other tasks. The point can be expected to be clarified in future studies. In this study, the DCD group was classified using MABC-2, an assessment battery for children. Our participants were recruited from a non-clinical population and were not diagnosed as DCD. Thus, our findings may not reflect the characteristics of clinical populations with DCD. However, the performance of executive function tasks was similar between children with DCD and those with motor clumsiness but no diagnosis of DCD (Leonard et al., 2015), which implied

that our results have application to clinical populations with DCD. We expect that our results will be confirmed by future studies with clinical populations.

5. Conclusion

We examined the association between motor clumsiness of DCD and ERP components in a Go/Nogo task. The results indicated that Nogo-P3 for the rare stimulus was smaller in the DCD group than in the control group, whereas there were no differences in Nogo-N2 between the groups. These findings indicate that the influence of motor clumsiness is limited to cognitive processes after the initiation of the actual motor response in the Go/Nogo task.

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Figure Legends

Figure 1. The Go/Nogo task

Figure 2. Grand averaged waveforms of event related potentials in the control group and the developmental coordination disorder (DCD) group.

Figure 3. Topographical maps of event related potential components

Table 1. Characteristics of the control group and the developmental coordination disorder (DCD) group

		control	DCD	P value
MABC-2 (SS)	M	6.12	9.93	<.001[a]
	SD	1.05	1.82	
	Min	3	8	
	Max	7	16	
Sex (n)	Male	24	11	1.00[b]
	Female	32	14	
Age (year)	M	23.41	24.48	.33[a]
	SD	4.51	5.33	
Handedness (n)	Right	54	24	1.00 [b]
	Left	2	1	
ADHD trait	M	30.61	32.44	.63 [a]
	SD	17.03	12.72	

ADHD trait = score of Wender Utah Rating Scale

a: Two-sample t-test , b: Fisher exact test

Table 2. Behavioral results

		Rare Nogo condition		Frequent Nogo condition	
		Control	DCD	Control	DCD
Correct reaction time (ms)	M	*303.46*	*305.82*	*383.58*	*368.26*
	SD	46.03	44.80	53.71	42.22
Omission error rate (%)	M	0.33	0.58	0.61	0.25
	SD	0.92	1.42	1.54	0.74
Commission error rate (%)	M	***7.25***	***11.19***	*0.09*	*0.06*
	SD	6.89	7.82	0.43	0.15

DCD = developmental coordination disorder group, *M* = mean, *SD* = standard deviation

Italic fonts represent differences between conditions (rare Nogo vs frequent Nogo; $p < .05$); **Bold fonts** represent differences between groups (control vs DCD, $p < .05$).

Table 3. Amplitudes (μ V) of event related potential components

			Rare		Frequent	
			control	DCD	control	DCD
Nogo-N2	Fz	*M*	-3.04	-2.87	-1.38	-1.66
		SD	2.57	2.05	1.46	1.72
	Cz	*M*	-2.35	-2.78	-0.35	-0.48
		SD	2.99	2.66	1.82	1.49
Nogo-P3	Fz	*M*	**0.84**	**0.04**	-0.90	-0.85
		SD	**2.90**	**2.77**	1.63	1.28
	Cz	*M*	**4.71**	**3.07**	-0.18	0.02
		SD	**3.55**	**2.81**	1.91	1.40
	Pz	*M*	**3.35**	**2.22**	0.06	0.03
		SD	**2.42**	**1.87**	1.17	0.97
Go-N2	Fz	*M*	-2.96	-2.22	-2.91	-3.38
		SD	2.54	2.21	1.90	2.59
	Cz	*M*	-1.65	-1.61	-0.73	-1.52
		SD	2.59	1.96	2.29	1.90
Go-P3	Fz	*M*	-1.68	-1.11	-1.23	-1.88
		SD	2.95	4.41	1.96	2.65
	Cz	*M*	2.02	1.66	1.66	0.84
		SD	3.37	3.58	2.56	1.87
	Pz	*M*	5.00	4.18	1.62	1.31
		SD	2.34	2.04	1.46	1.55

DCD = developmental coordination disorder group, *M* = mean, *SD* = standard deviation

Bold fonts represent differences between groups (control vs DCD, $p < .05$).

Highlights

Nogo-P3 was reduced in the developmental coordination disorder (DCD) group.

There were no differences in Nogo-N2 between DCD and control groups.

The influence of DCD is limited to process after starting the motor response.

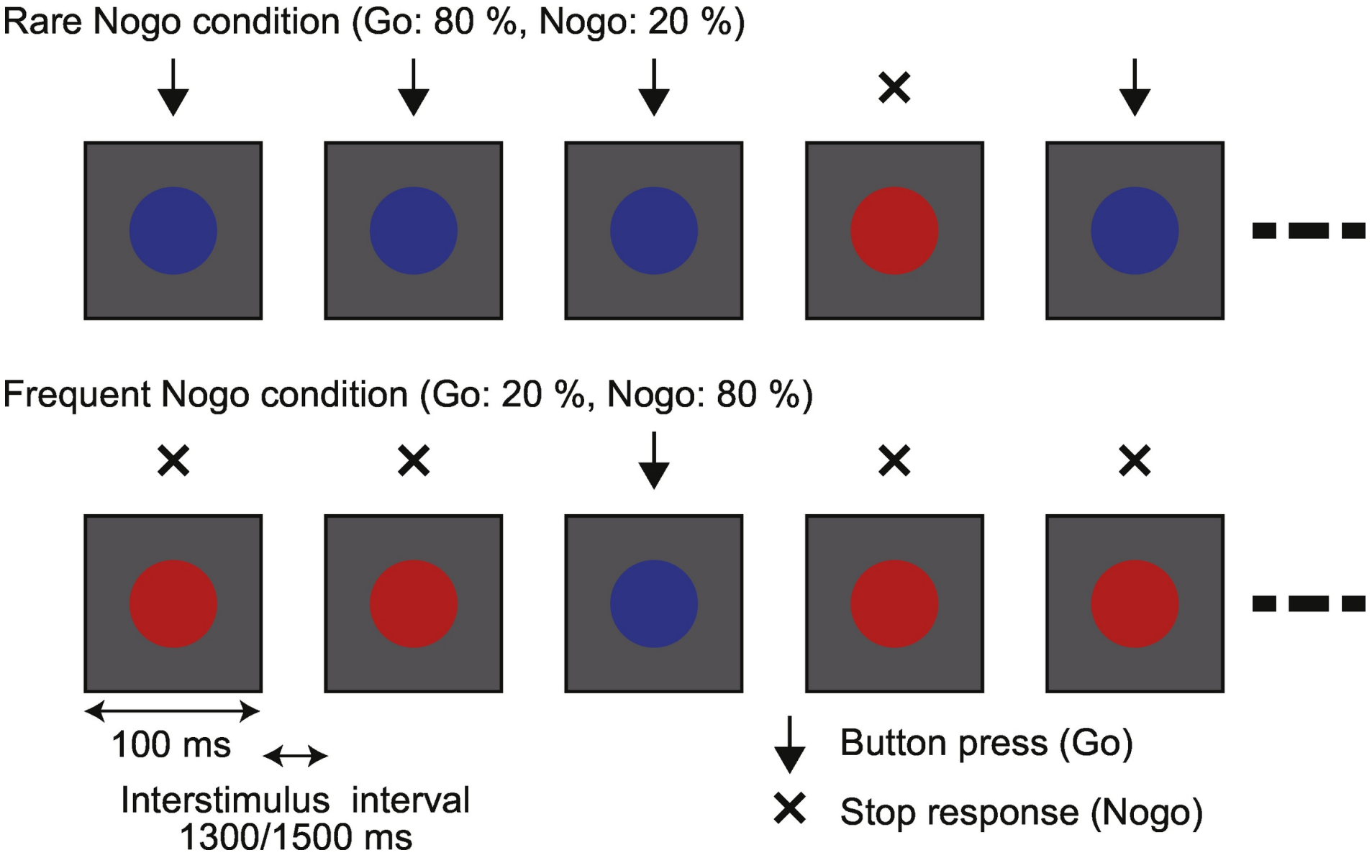
Rare Nogo condition (Go: 80 %, Nogo: 20 %)
Frequent Nogo condition (Go: 20 %, Nogo: 80 %)
100 ms
Interstimulus interval
1300/1500 ms
Button press (Go)
Stop response (Nogo)

Figure 1

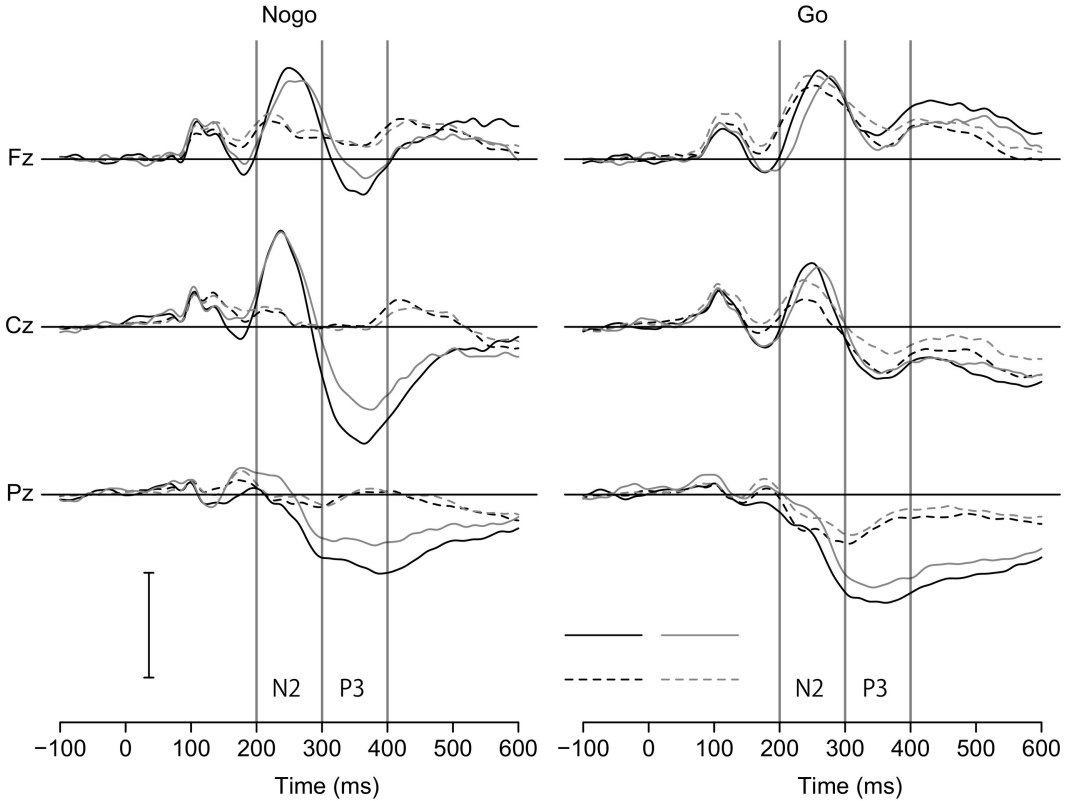
Nogo
Go
Fz
Cz
Pz
N2
P3
N2
P3
−100
0
100
200
300
400
500
600
Time (ms)
−100
0
100
200
300
400
500
600
Time (ms)

Figure 2

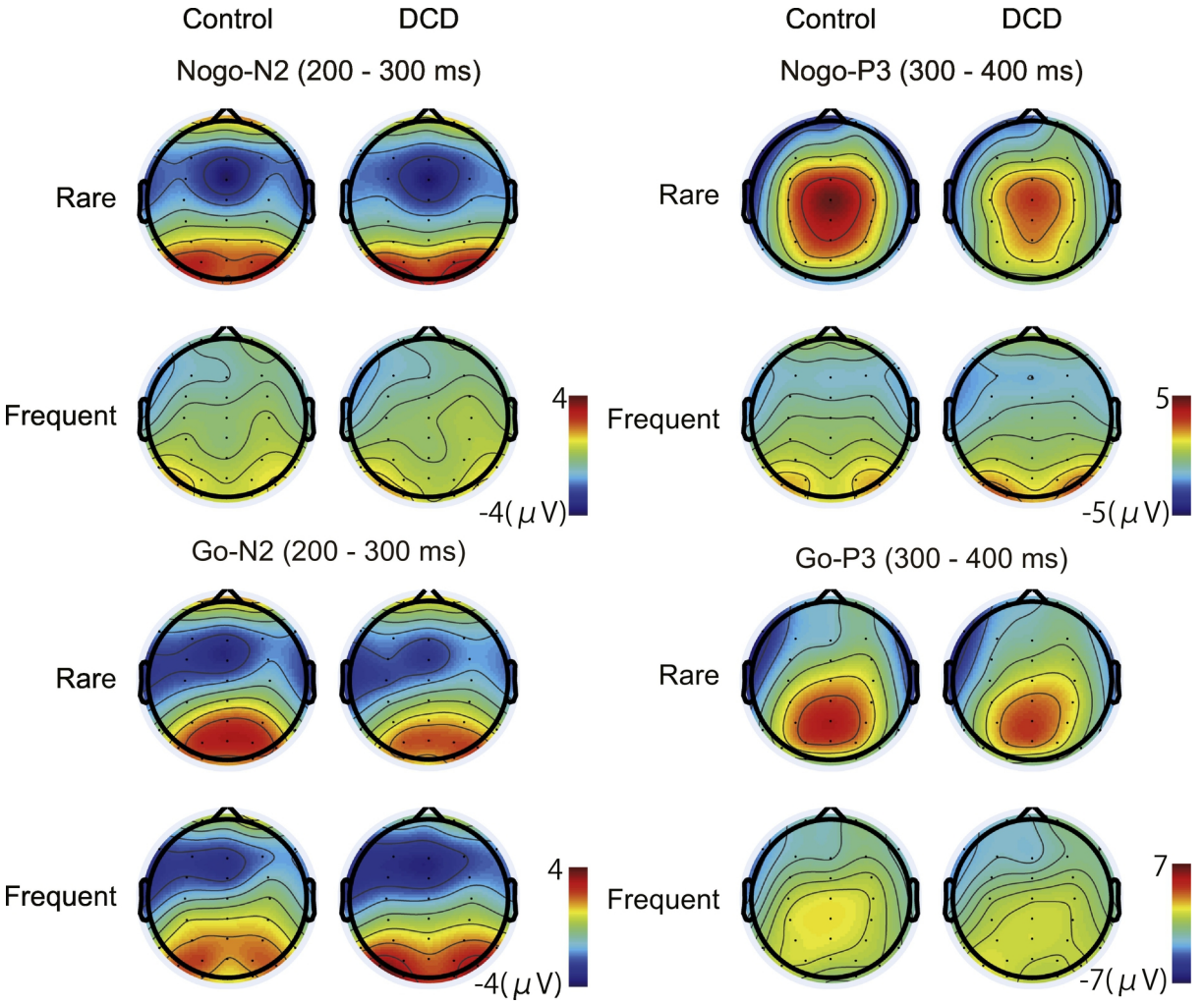
Control
DCD
Nogo-N2 (200 - 300 ms)
Rare
Frequent
4
-4(μV)
Control
DCD
Nogo-P3 (300 - 400 ms)
Rare
Frequent
5
-5(μV)
Go-N2 (200 - 300 ms)
Rare
Frequent
4
-4(μV)
Go-P3 (300 - 400 ms)
Rare
Frequent
7
-7(μV)

Figure 3